

# Aneurysm Rupture after EVAR: Can the Ultimate Failure be Predicted?

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## KEYWORDS

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aneurysm repair;  
EVAR;  
Prognosis

**Abstract** *Objectives:* To provide insight into the causes and timing of AAA rupture after EVAR.

*Design:* Original data regarding AAA ruptures following EVAR were collected from MEDLINE and EMBASE databases. Data were extracted systematically and patient and procedural characteristics were analyzed.

*Results:* 270 patients with AAA ruptures after EVAR were identified. Causes of rupture included endoleaks (in 160: type IA 57, type IB 31, type II 23, type III 26, type IV 0, endotension 9, unspecified 14), graft migration 41, graft disconnection 11 and infection 6. Most of the described AAA ruptures occurred within 2–3 years after EVAR. Mean initial AAA diameter was relatively large (65 mm). No abnormalities were present in 41 patients during follow-up before rupture. Structural graft failure was described in 96 and a fatal course in 119 patients. *Conclusions:* Focus of surveillance on the first 2–3 years after EVAR may possibly reduce the AAA rupture rate, especially in patients with increased risk of early rupture (relatively large initial AAA diameter or presence of endoleak or graft migration). Better stent-graft durability and longevity is required to further reduce the AAA rupture risk after EVAR. Complete prevention will however remain challenging since AAA rupture may occur even if no predisposing abnormalities are present.

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## Introduction

The frontier of endovascular aortic repair has progressed rapidly during the last 15 years. With increasing incidences of detected aortic aneurysms due to screening, improvements of detection methods and increasing age of people,<sup>1,2</sup> as well as a rapidly expanding group of vascular specialists trained in endovascular techniques, the rate of endovascular abdominal aortic aneurysm repair (EVAR) is expected to further increase in the future.

The only real goal of EVAR is to prevent aneurysm rupture. Nonetheless, rupture still occurs. Large cohort studies have reported rupture rates between 0.5 and 1.2% per patient per year after EVAR.<sup>3–8</sup> Life time risks are even higher, because most patients live for several years after the procedure.<sup>9</sup> AAA rupture is therefore a small but significant risk after EVAR and a major limiting factor in the prognosis of patients who undergo this minimally invasive procedure.

In order to improve the prognosis of patients after EVAR, more insight into the causes and timing of AAA rupture after EVAR is necessary. The purpose of this study is to investigate the causes and timing of rupture after EVAR by means of a review of all available data regarding AAA rupture after EVAR.

## Materials and Methods

### Literature search

MEDLINE, EMBASE and Cochrane library CENTRAL databases were searched up to March 1, 2008. The following search string was used for Medline: (AA[Title/Abstract] OR "aortic aneurysm"[Title/Abstract] OR "aneurysm aortic"[Title/Abstract] OR ("aneurysm"[Title/Abstract] AND "aorta"[Title/Abstract]) OR ("aneurysms"[Title/Abstract] AND "aorta"[Title/Abstract]) OR "AAA"[Title/Abstract] OR "TAAA"[Title/Abstract] OR "TAA"[Title/Abstract] OR "aorta, thoracic"[MeSH Terms] OR "Aortic Aneurysm, Thoracic"[MeSH Terms] OR "aortic aneurysm, abdominal"[MeSH Terms]) AND (ruptur\*[Title/Abstract] OR dissect\*[Title/Abstract]) AND (endovasc\*[Title/Abstract] OR endolum\*[Title/Abstract] OR intralum\*[Title/Abstract] OR EVAR[Title/Abstract] OR TEVAR[Title/Abstract] OR stent\*[Title/Abstract] OR endograf\*[Title/Abstract]). This resulted in 1720 articles.

The following search string was used for Embase: ((abdominal AND aortic AND 'aneurysm'/syn) OR (thoracic AND aortic AND 'aneurysm'/syn) OR ('thorax'/syn AND 'aorta'/syn AND 'aneurysm'/syn) OR ('abdomen'/syn AND 'aorta'/syn AND 'aneurysm'/syn)) AND ('rupture'/syn OR 'dissection'/syn) AND ('endovascular'/syn OR 'endoluminal'/syn OR 'intraluminal'/syn OR 'EVAR'/syn OR 'TEVAR'/syn OR 'stent'/syn OR 'endograft'/syn OR 'graft'/syn) AND [embase]/lim. This resulted in 2030 articles. Cochrane library CENTRAL database was browsed manually and did not result in any relevant articles. After removal of duplicate articles, 2794 unique articles remained.

### Selection of articles

All titles and abstracts of the selected articles were read by two independent investigators (F.J.S. and B.E.M.). Inclusion

criteria were a) domain of the study consisted of patients with abdominal aortic aneurysms, b) EVAR was performed, c) AAA rupture after initiation of EVAR was described for one or more patients and d) original data were presented about one or more pre-defined variables for these patient(s) with AAA rupture after EVAR. No patients with thoracic aortic aneurysms were included. Full-text versions were obtained of all articles that matched the inclusion criteria and were subsequently read by two independent investigators (F.J.S. and B.E.M.). No language or publication date restrictions were applied. Reference lists were searched manually to locate additional relevant articles. This resulted in a final selection of 110 articles.

### Data extraction

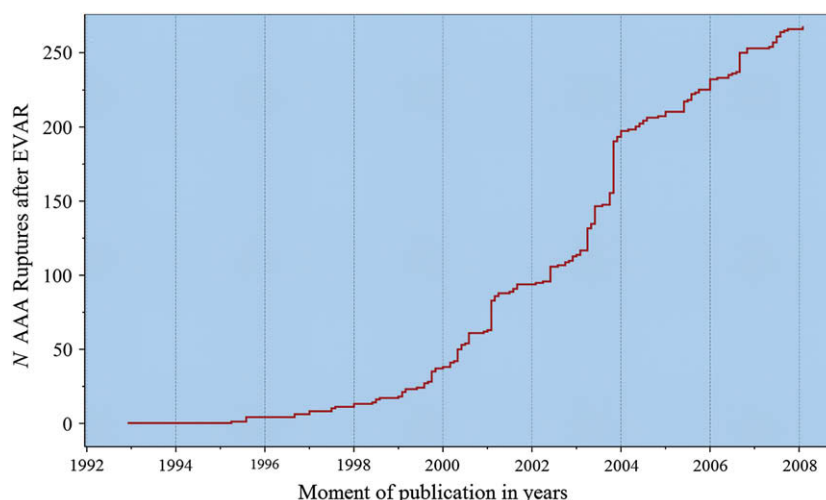
The following characteristics were extracted from articles that explicitly presented original data about patients with aneurysm rupture after EVAR: patient characteristics (age, sex), aneurysmal characteristics (location, diameter, proximal neck diameter and length), stent-graft characteristics (diameter, length, brand, type), aneurysm exclusion at the initial post-operative angiography, time interval between EVAR and aneurysm rupture, time interval between most recent follow-up visit and aneurysm rupture, indication and success of re-interventions during follow-up, aneurysm diameter changes (defined by  $\geq 5$  mm change or more), detection of endoleaks during follow-up (type IA, type IB, type II, type III, type IV or endotension), other complications during follow-up (including graft migration, wire fractures, angulation, insecure fixation, aneurysm inflammation), adherence to follow-up schedule, refusal of recommended re-interventions, symptoms at the time of aneurysm rupture, diagnostic examinations, main cause of aneurysm rupture as described by the authors of the report, graft material failure (including migration, disconnection, fabric perforation, broken sutures, wire breakages, stent erosion, type III endoleak), open or endovascular rupture repair, complications of aneurysm rupture repair and publication dates. Other articles written by identical authors and/or institutions were studied in detail and excluded if necessary to prevent multiple inclusions of the same patients. All extracted information was entered systematically in a database. Totally, original data about 270 patients with AAA ruptures after EVAR were identified.

### Statistical analysis

Articles were grouped in quartiles according to publication date to search for differences in variables between early and more recently reported aneurysm ruptures. Data were analyzed with SPSS version 14.0. Student's *t*-test was performed for comparison of means. A *P*-value less than 0.05 was considered significant.

## Results

Original data about 270 patients with aneurysm ruptures after EVAR were identified. Fig. 1 represents the distribution of the AAA ruptures after EVAR by publication date of



**Figure 1** Patients with AAA rupture after aneurysm repair by publication date. AAA, abdominal aortic aneurysm.

the article that described the rupture. An acceleration of reports can be seen between 2000 and 2004 and this number declined during recent years. Table 1 shows the availability of extracted data per studied variable. Data regarding moment, cause and fatality of the rupture were reported in almost all patients, while information about more detailed variables, such as aneurysm neck characteristics and graft dimensions, was only available for smaller groups of patients.

### Moment of AAA rupture

Data about the moment of rupture were available for 202 patients. AAA rupture occurred in 38 of these patients during the perioperative period ( $\leq 30$  days). The AAA rupture occurred in the other 164 patients during follow-up and the mean time interval between the initial procedure and subsequent AAA rupture of these patients was  $24 \pm 18$  m (range 2.0–96.0 m, median 20 m).

### Patient characteristics

Information about age was available for 76 patients with AAA ruptures after EVAR. The mean age of these patients was  $74 \pm 8.6$  y at the moment of AAA rupture (range 56–93 y). 70 of the 76 patients for which information about gender was available were male. Mean age at the moment of rupture was 74 y for both men and women.

### Aneurysm characteristics

For 8 patients was explicitly stated that the initial EVAR was an emergent procedure for ruptured AAA ( $N = 8$ ). Information about the initial AAA diameter was available in 94 patients (Fig. 2). Mean initial AAA diameter of these patients was  $65 \pm 14$  mm. Deployment of an aortouniiliac stent graft during the initial EVAR was described for 13 patients. Data about other aneurysm characteristics, such as proximal neck length, diameter, angulation, iliac diameters or tortuosity, were unfortunately reported scarcely.

### Perioperative ruptures

38 of the AAA ruptures occurred in the perioperative period ( $\leq 30$  d), of which 13 during the initial procedure and 25 after the procedure but within 30 days after the procedure. Characteristics of the 13 patients with AAA ruptures during the initial EVAR are shown in Table 2. Grouping of patients in quartiles according to publication date revealed that 28 of the 38 perioperative ruptures occurred in Quartiles 1 and 2 and only 10 of the 38 in the more recent Quartiles 3 and 4. In 10 of the 25 aneurysms that ruptured after EVAR within 30 days was explicitly stated that the aneurysm was not completely excluded after the initial procedure (due to proximal type I leak  $N = 7$ ; distal type I leak  $N = 2$ ; limb maldeployment  $N = 1$ ). A fatal course was described for half of the patients with perioperative ruptures ( $N = 19$ ).

### Late ruptures

Fig. 3 shows the distribution of the time periods between EVAR and subsequent AAA ruptures. The mean time interval between the initial procedure and subsequent AAA rupture was 15 m, 24 m, 28 m and 30 m for publication date quartiles 1, 2, 3 and 4, respectively. 155 of the 202 described moments of rupture were within 27 months after EVAR and the number of ruptures strongly declined thereafter. The mean time interval did not differ between men and women for the 71 patients in which information about both gender and time interval was available.

In 82 patients, information about both time interval and initial AAA diameter was available. Patients with an initial AAA diameter above mean ( $>65$  mm) had a shorter time interval between EVAR and the AAA rupture than patients with AAA diameters  $\leq 65$  mm, although this difference was not significant (22 m vs. 29 m,  $P = 0.09$ , 95% CI of difference  $-1.0$  to 15 m).

### Symptoms of aneurysm rupture after EVAR

Information about symptoms of the patients was available for 71 patients. The most common symptoms included

**Table 1** Availability of information for data extraction. Presented are the number and percentage of patients with aneurysm ruptures for which the article explicitly described information about the pre-defined variable

Variable	Data explicitly stated	
	N	Reference numbers in <a href="#">supplement</a>
Main cause of rupture	235	1, 2, 4–21, 23, 25–44, 46–50, 52–70, 72–83, 85–110
Fatality of the rupture	231	1–5, 7–29, 31, 32, 34–39, 41–44, 46–60, 63–69, 71–85, 87–92, 94–108, 110
Performance of rupture repair	210	2, 4–6, 7, 8, 11–17, 20–23, 25, 27–32, 34, 36–39, 41, 43–48, 50–55, 57–61, 63–69, 71, 73–76, 78–91, 94, 95, 97–101, 103, 110
Time interval between initial AAA repair and rupture	202	1–6, 8–16, 18, 20–25, 27–29, 31–34, 37–39, 42–78, 80–92, 94–100, 102–105, 107, 108, 110
Follow-up findings <sup>a</sup>	145	1, 2, 5, 10–13, 16, 18–20, 22, 23, 25, 27, 29, 31–33, 35–38, 41–43, 46, 48, 49, 52–55, 60–65, 67, 70, 73, 74, 76, 78, 80, 82, 84, 85, 87–91, 94, 96–99, 103–108
Graft type	133	1, 2, 5, 6, 8, 10, 12–14, 16–19, 21, 23, 25, 27, 28, 31–39, 42–44, 46–50, 52, 54, 55, 57, 58, 61, 63, 64, 67–71, 73–76, 78, 80, 81, 84–86, 88–91, 93, 94, 97–100, 102–108, 110
Presence of complications after rupture repair	129	2, 4, 5, 7, 8, 11–14, 16, 17, 20, 21, 23, 24, 27–29, 31, 32, 34, 37–39, 43, 44, 46–48, 50–55, 57, 58, 60, 63–69, 71, 73–76, 78–81, 83–85, 87–91, 94, 95, 97–99, 101, 103–108, 110
Sac shrinkage or enlargement	101	1, 2, 5, 12, 16, 18, 22–25, 27–29, 31–33, 35, 38, 43, 44, 46, 49, 52–55, 60–63, 65, 67, 70, 73, 74, 76, 78, 80, 82, 84, 85, 87–91, 97, 99, 103, 104, 106, 107
Graft material failure	97	2, 4, 6, 7, 10, 11, 14, 17, 23, 25, 27, 28, 32, 34, 38, 44, 46, 48, 52, 54, 55, 57, 58, 60, 62, 65, 67–70, 72, 74, 76, 78–80, 82, 84–86, 88–91, 99, 103, 104, 106, 108, 109
Initial AAA diameter	94	2, 5, 8, 16, 17, 19, 23, 27, 29, 31, 32, 34, 38, 39, 46, 48, 50, 52, 54, 57, 61, 63–65, 67, 71, 73, 74, 76, 78, 80, 81, 83, 85, 88–91, 94, 97, 99, 100, 103, 104, 107, 108, 110
Gender	81	1, 2, 4–6, 8–13, 16–19, 23, 26, 28, 31, 34, 36, 39, 46, 48, 50, 52, 54, 57, 62–64, 71, 73, 74, 76, 78, 80–83, 85, 88–92, 94, 97, 99, 100, 102–108, 110
Age <sup>b</sup>	76	1, 2, 4, 6, 8, 9, 11, 12, 16–19, 22, 23, 26, 31, 34, 39, 43, 46, 48, 50, 52, 54, 57, 61, 63, 64, 71, 73, 74, 76, 78, 80, 81, 83–85, 88–91, 94, 97, 99, 100, 103–108, 110
Symptoms of rupture	71	1, 2, 4, 5, 8, 12, 16, 17, 23, 31, 34, 37, 43, 46, 48, 50, 52, 53, 55, 57, 60, 63–65, 68, 71, 73, 74, 76, 78–83, 85, 88–91, 97, 100, 103–108
Adherence to follow-up schedule	45	5, 11, 13, 17, 19, 32, 33, 35–38, 42, 48, 54, 60, 64, 67, 70, 71, 74, 76, 78, 80, 97, 105–108
Initial post-EVAR angiography	34	8, 12, 13, 17, 18, 23, 26, 31, 34, 48, 50, 55, 57, 63, 64, 71, 73, 76, 78, 80, 84, 85, 88–90, 93, 96–98, 100, 103, 108
Moment of initial EVAR procedure	23	11, 17, 48, 52, 54, 57, 64, 78, 88–91, 97, 99, 100, 103, 104, 108
Length of initial proximal aneurysm neck	17	19, 23, 31, 46, 50, 54, 57, 63, 64, 74, 76, 89, 90, 97, 100, 108
Diameter of initial proximal aneurysm neck	14	19, 23, 27, 31, 46, 50, 57, 61, 64, 89, 97, 100
Graft diameter	12	1–3, 5, 72, 80, 93, 104, 110
Graft length	9	1–5, 72, 76, 93, 104

<sup>a</sup> Follow-up findings include endoleaks, graft migration, wire fractures, angulation, rupture detection during regular follow-up visit, insecure fixation, signs of aneurysm inflammation.

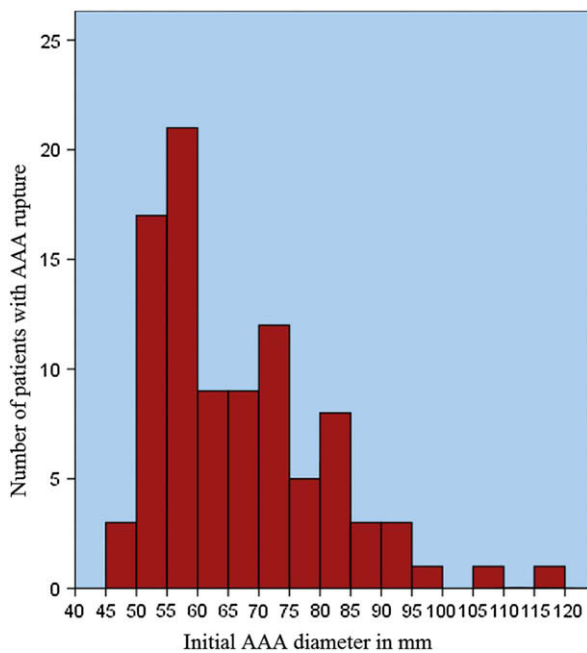
<sup>b</sup> Age at moment of rupture.

abdominal pain in 40, back pain in 33, hypovolemia/hypotension or shock in 23 and collapse or syncope in 12 patients.

### Causes of AAA ruptures after EVAR

The main cause of rupture was described for 235 of the 270 patients. [Fig. 4](#) shows these causes stratified by quartile of

publication date. Endoleaks were described as the main cause of rupture in 160 of the 235 patients. [Fig. 5](#) shows the share of different types of endoleaks in patients with AAA rupture after EVAR due to endoleak. Endoleak type IA (proximal attachment site endoleak) caused AAA rupture in 57, endoleak type IB (distal attachment site endoleak) in 31, endoleak type II (retrograde filling from lumbar arteries



**Figure 2** Distribution of initial AAA diameters.  $N = 93$ . AAA, abdominal aortic aneurysm. Distribution of the initial maximal AAA diameters before EVAR was initiated. One patient with an aorto-iliac aneurysm with an iliac component measuring 64 mm and aortic component measuring 33 mm is not included in this graph.

and/or IMA, sacral, gonadal or accessory renal artery) in 23, endoleak type III (structural failure of the device) in 26, type IV (fabric porosity) in 0, and endotension in 9 (continued expansion in the absence of a confirmed

endoleak, type V endoleak). Endoleak type was not specified in 14 of the patients with rupture due to endoleak. The type of endoleak was determined after rupture had occurred in most patients on the basis of CTA findings or findings during open surgical conversion. Presence of endoleak at the last follow-up moment before rupture was described for 56 patients in which the main cause of rupture was endoleak.

Graft migration was responsible for 41 ruptures and graft disconnection for 11 ruptures. Inflammation or infection of the AAA caused rupture in 6 patients. Less frequent causes included rupture of an iliac component of the aneurysm ( $N = 3$ ), rupture after initial EVAR because left graft limb could not be deployed during initial procedure ( $N = 1$ ) and rupture during thrombolysis because of stent-graft occlusion ( $N = 1$ ). When comparing patients by quartile of publication date, a decreasing trend of graft migrations, disconnections and perioperative ruptures was noted, while the share of endoleaks appeared to increase during time (Fig. 4). Structural failure of graft material, as defined by fabric perforation, broken sutures, wire breakages, graft migration, graft disconnection, graft erosion or type III endoleak, was explicitly mentioned in 96 of all ruptures.

### Follow-up

Refusal of one or more follow-up visits before the rupture occurred was explicitly mentioned for 43 AAA ruptures after EVAR. Additional refusal of a recommended re-intervention was explicitly mentioned in 18 of these patients. The refusal of the follow-up or re-intervention was a "patient's decision" in all of these patients, except in 3 cases in which the decision was made by the treating physician because of severe comorbidities of the patient.

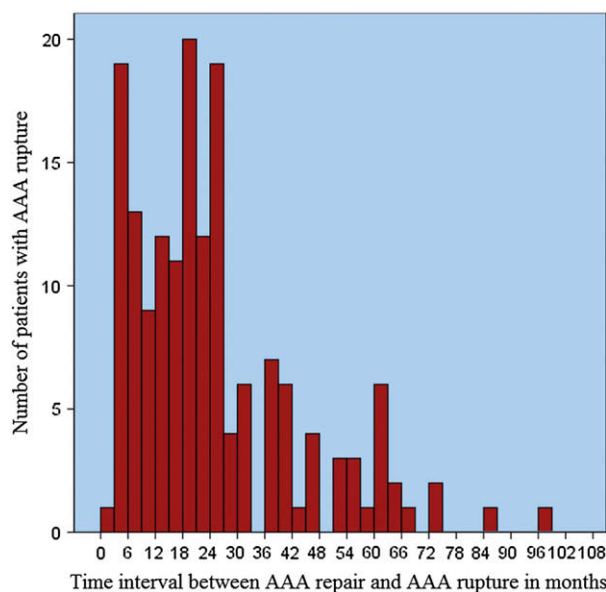
**Table 2** Intra-operative ruptures

No.	Graft	Cause	Repair	Mortality
1	NR	NR	NR	+
2	NR	NR	Open conversion	NR
3	NR	NR	Open conversion	NR
4	AneuRx	Balloon dilatation of proximal aortic neck	Open conversion	NR
5	AneuRx	Instrumentation and perforation of aneurysm sac	Open conversion	NR
6	Nottingham system	Rupture during attempts to remove premature deployed proximal stent	Open, axillobifemoral bypass	+
7	Nottingham system	Aortic neck rupture	Open conversion	+
8	Nottingham system	Common iliac rupture close to bifurcation during advancement of graft because of iliac tortuosity	Open conversion	—
9	NR	Balloon dilatation of proximal aortic neck	Open conversion	—
10	Vanguard	Iatrogenic	Endovascular <sup>a</sup>	—
11	NR	Directly after stent-deployment	Open conversion	—
12	Z-stents, PTFE graft	Directly after balloon dilatation	Open conversion	—
13	White—Yu tube	Rupture of proximal neck during deployment of endograft by balloon inflation	Open conversion	+

NR = Not reported.

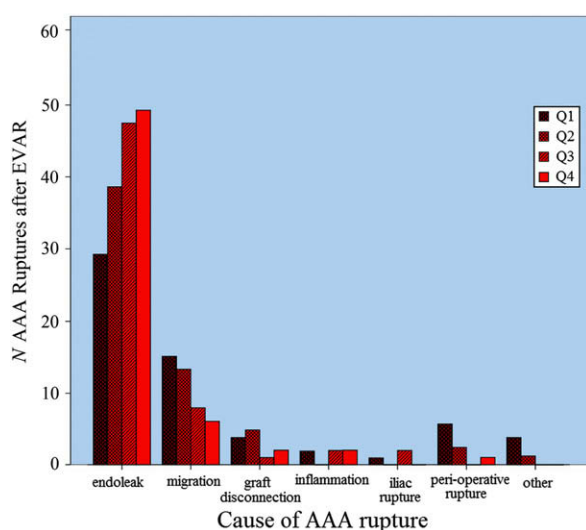
<sup>a</sup> Immediate graft deployment.





**Figure 3** Time interval between endovascular AAA repair and subsequent AAA rupture.  $N = 164$ . AAA, abdominal aortic aneurysm. All AAA ruptures are selected that occurred after the perioperative period (after initial 30 days).

A description of the course of AAA sac diameter during follow-up was presented in 101 patients. Enlargement of the AAA sac during follow-up occurred in 36 of these patients, no change in 39 and shrinkage in 26 patients. For 122 patients, the presence of other follow-up findings was explicitly stated. In 35 patients no abnormalities were found during follow-up (absence of endoleak, wire fractures, migration, graft angulation, insecure fixation, signs of inflammation and sac enlargement). In another 6 patients only a small type II endoleak was found during follow-up and the AAA sac was stable or shrunken. So, in 41



**Figure 4** Main causes of AAA rupture after EVAR by quartiles of publication date.  $N = 235$ . AAA, abdominal aortic aneurysm; Q, quartile. Q1 contains the first descriptions and Q4 the most recent descriptions of AAA ruptures after EVAR.

patients no abnormalities were found during follow-up that required re-intervention.

Endoleaks were described for 56 of all patients during the most recent follow-up visit before the rupture took place, of which 6 small type II endoleaks without sac enlargement. Other abnormal follow-up findings included wire fractures, graft migration or severe graft angulation in 11, insecure fixation in 5, AAA sac inflammation in 1, and, interestingly, the AAA rupture itself was diagnosed during a regular follow-up visit in 5 patients with AAA ruptures after EVAR.

### Repair and outcome

AAA rupture repair was performed in 160 patients by open conversion, in 26 with endovascular techniques, in 24 no repair was performed at all and in 60 patients no data were available about performance of rupture repair.

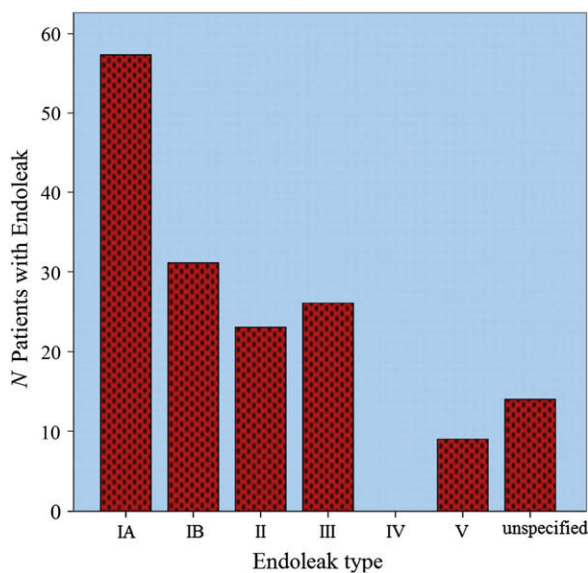
AAA ruptures were fatal in 119 of 231 patients for whom data were available about mortality. In patients who underwent endovascular or open repair of the AAA rupture after EVAR, the outcome was slightly better: a fatal course in 62 of 138 patients that underwent open ruptured AAA repair, and in 7 of 26 patients that underwent endovascular ruptured AAA repair.

### Discussion

Original data about 270 patients with AAA ruptures after EVAR were identified. Most of the described AAA ruptures after EVAR occurred within the first 2–3 years of follow-up. The mean initial AAA diameter of patients with ruptures after EVAR was relatively large: 65 mm. Most common symptoms included abdominal pain, back pain and signs of hypovolemia. Endoleaks were by far the most frequent cause of AAA rupture and graft migration the second most frequent cause. A decreasing trend of graft migrations, disconnections and perioperative ruptures was noted during time, while the share of endoleaks appeared to increase with moment of publication. Refusal of follow-up or re-intervention was reported for 43 of the 270 patients with AAA ruptures after EVAR, most of these due to a "patient's decision". Rupture repair was performed in a majority of patients and open conversion was by far the most common approach. Overall, AAA ruptures were fatal in approximately half of all patients. Structural failure of the graft material was reported for 96 of the 270 patients.

Because AAA rupture after EVAR is a relatively rare event and patients will not always present their AAA rupture in the same institution, the collection of original data about a large number of patients with AAA ruptures is very difficult for AAA registries and even harder for single institutions or authors. The largest group of patients with AAA ruptures after EVAR that has been presented before consisted of 34 patients from the EUROSTAR registry.<sup>10</sup> The presented methodology, a review of all original, published data about patients with AAA ruptures after EVAR, led to new opportunities in our struggle to improve insight into this devastating complication after EVAR.

The presented approach has however some limitations. Because data collection was restricted to information that was available in existing literature, information about many



**Figure 5** Specification of type of endoleak of patients with AAA rupture after EVAR due to endoleak. AAA, abdominal aortic aneurysm.

variables could not be retrieved for all patients. For example: data about the aneurysm neck diameter at the moment of EVAR and at the time of rupture were lacking, while this information may be very important because the aneurysm neck size may play a major role in mechanisms of graft migration and proximal endoleak. The presented results should be interpreted carefully and most presented values may be best regarded as minimum values of a range, where the 'true' value is likely to reside. Information about the year of repair was only available for 23 of the 270 patients, which unfortunately did not allow valid comparison of patients by year of AAA repair. Although use of the publication date variable has its limitations, it was probably the best available tool to search for differences between device generations in this specific type of study. In addition, if old series with old devices have been published later, then the presented differences will only be an underestimate of the real differences between early and later generation stent grafts, because the first quartile probably consists of early generation stent grafts only, while the last quartile is more likely to be a mix of early and later generation stent grafts.

Another disadvantage of this study, which is inherently linked to the chosen approach, is that selective publication of patients with AAA rupture after EVAR may have lead to publication bias. Publication bias is the phenomenon in which positive results have a better chance of being published than negative results. Articles about patients who survived AAA rupture after EVAR may possibly have a different chance of publication than articles about patients who did not survive AAA rupture after EVAR.

Most aneurysm ruptures in our cohort occurred within 2–3 years. Data about many patients with AAA rupture after EVAR were presented in case reports or case series which were not limited by a specific follow-up duration (39 of the 110 articles) or were presented in cohort studies with long-term follow-up. A significant number of patients

with rupture after EVAR were however presented in cohort studies with limited follow-up duration. This may partially explain the relatively high number of AAA ruptures in the first 2–3 years after EVAR that were described in the original articles. Ruptures after 3 years may also be slightly underrepresented in our cohort due to movement of patients to other institutions and nursing homes, or due to mortality.<sup>9</sup>

Additionally, every rupture after EVAR is a rare case and may offer sufficient new information for publication. Selection bias due to selective publication of the AAA ruptures after EVAR is however definitely important to consider and to be aware of when the presented results are interpreted. The precise effect of potentially present publication bias on the selection of patients is hard to determine. From our point of view, the possibility of publication bias should be kept in mind, but the effect of this type of bias is probably less than moderate in the context of this topic. As a corollary, there may be bias against publishing case reports of rupture after EVAR; for example, as migration reports became well established in the literature, publication of additional cases becomes increasingly difficult.

Another disadvantage of the study is that determination of the cause of aneurysm rupture was based on the description of the cause of the rupture by the authors of the selected articles. Because different authors of different articles may have used different criteria for the determination of the cause of rupture, this process may have been relatively subjective. The most important cause of rupture was determined by the authors of the selected articles, and although it sometimes may have been subjective, it is probably the best estimation of the cause of the aneurysm ruptures in this type of study.

Because data collection was restricted to information that was presented in the original articles, diagnosis of AAA rupture after EVAR was determined by the description of the diagnosis by the authors of the identified articles. In 24 of the 270 patients, no precise description of the performed diagnostic examinations was provided and no fatal course was described. Theoretically, there may have been a possibility that any of these 24 patients had a "symptomatic" AAA instead of a ruptured AAA. The diagnosis was determined by the authors of the selected articles, and although it sometimes may have been subjective, it is probably the best estimation of diagnosis of AAA rupture after EVAR for this specific type of study.

A significant association between larger aneurysm size and increased rupture risk after EVAR has been described by several investigators, including the EUROSTAR Registry, Lifeline Registry and AneuRx trial.<sup>1–14</sup> The EUROSTAR investigators also demonstrated that endoleak type I and III and device migration are significantly associated with rupture risk after EVAR.<sup>14,15</sup> Increased rupture risk in patients with persistent type II endoleak (>6 months) was described by Jones et al.<sup>16</sup> Female gender may increase the rupture risk too.<sup>12,13</sup> No differences between patients aged ≥80 y or patients <80 y were found in the EUROSTAR Registry.<sup>17</sup>

The findings in the current study may have several important implications for clinicians and policymakers. Focus of surveillance after EVAR to the first 2–3 years may

possibly improve the efficiency of follow-up after EVAR. Relatively many ruptures occur between the follow-up visits at one and two years after EVAR. An additional follow-up moment after 18 months may therefore possibly reduce the AAA rupture rate. This may be especially important for patients with an increased risk for a relatively "early" rupture, such as patients with large initial AAA diameters or with endoleaks or graft migration. Because refusal of follow-up and refusal of re-interventions were relatively prevalent in the presented group of patients with AAA ruptures, more attention should be paid in the communication with patients before and after EVAR to the importance of follow-up adherence and performance of required re-interventions. An informed consent may also need to mention and acknowledge the small but continued risk of aneurysm rupture after EVAR. Better sustainability of stent-graft material is required to further reduce the risk of AAA rupture after EVAR. Purpose of future research should be improving the efficiency of the follow-up schedule after EVAR and evaluation of new stent-graft designs to prevent graft failure.

In conclusion, surveillance should focus on the first 2–3 years, especially in patients with increased risk of early rupture (relatively large initial AAA diameter or presence of endoleak or graft migration). Better stent-graft durability and longevity is required to further reduce the AAA rupture risk after EVAR. Complete prevention will remain challenging since rupture may occur even if no predisposing abnormalities are present. With some reasonable efforts, dramatic reductions in rupture rates may however become reality soon.

### Conflict of Interest Statement

No conflicts of interest, no study sponsors.

### Acknowledgements

No conflicts of interest declared.

### Appendix Supplementary data

Supplementary data associated with this article can be found, in the online version, at [doi:10.1016/j.ejvs.2008.10.011](https://doi.org/10.1016/j.ejvs.2008.10.011).

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